Even Studies of Hiroshima Have Not Proved Mutations

GENETIC DAMAGE is one of the most frightening of the potential side effects of the continued pollution of our environment. It is also the least well understood. It is a rare layman who realizes just what "genetic damage" might mean to his own health and survival, or his children's.

The expert has every reason to point the finger of suspicion at a great many potential hazards, but he also lacks proven criteria by which to translate scattered laboratory findings into reliable estimates of human risk.

Radiation was the first environmental factor proven, in the late H. J. Muller's experiments with fruit flies, to cause genetic damage, or as we might now say, to cause mutations of chemical changes in DNA. This mutagenic effect has been found in every one of many hundreds of biological systems tested, and no one doubts that human genes are also susceptible to radiation effects.

THE WHOLE subject became a matter of intense scientific and political debate in criticisms of nuclear weapons tests prior to the nuclear test ban treaty. Nevertheless, there is no direct way to prove that any gene mutations were ever produced in human germ cells (sperm or eggs) by an exposure to radiation.

Even the studies of the survivors of the bombings of Hiroshima and Nagasaki have given only marginal, inconclusive evidence of such effects. This is no surprise to geneticists who have calculated the expected level of changes from animal studies and who know how difficult it is to standardize observations on people.

Although the ultimate chemistry of radiation effects on DNA also remains obscure, radiation is also the easiest agent for such calculations. We do understand the physics of penetration of radiation energy into tissue, and we have little difficulty in calculating the doses that are delivered to the DNA of germ cells in various circumstances.

This does give us one standard of environmental health, namely, the level of radiation to which we are already exposed through the action of cosmic rays and the natural radio-activity of the earth. However, even these results could be minimized—if our eventual knowledge of their biological effects moved us to it—by shielding our bodies or homes, or more likely by taking advantage of new knowledge of chemical effects that stimulate the cells' capacity to repair part of the damage inflicted on DNA.

For example, it has been known for a long time that radiation had less effect on cells deprived of oxygen, and it has recently been indicated that oxygen inhibits DNA repair, perhaps by further chemical reaction with the damaged parts of the DNA molecule. One of the chief problems in translating the effects of chemical genetic poisons is the uncertainty about their penetration through the body and into germ cells. This also affects our reasoning about scaling from large doses, needed ot do convenient experiments, to small ones typical of large-scale human exposure.

For example, chlorine (as used in bleach or to sanitize drinking water) is undoubtedly capable of reacting with, modifying and destroying DNA. But we might reasonably hope that the small doses we habitually imbibe are neutralized by other body substances before they can do any genetic damage.

This is precisely a reasonable hope, lacking experimental evidence either way. The same holds true for many other environmental chemicals—especially ozone, peroxides (and smog?), formaldehyde and other gaseous disinfectants.

Before we can critically assess these particular chemicals, and agents like cyclamates or LSD, we should review different manifestations of genetic damage, as I propose to do in subsequent columns.